DOCUMENT RESUME

ED 232 983 SP 022 839

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TITLE The Influence of a 12-Week Conditioning Program on Growth Hormone and Somatomedin C Concentrations in Moderately Overweight Males.
SPONS AGENCY North Carolina State Univ., Raleigh.; North Carolina Univ., Greensboro. School of Home Economics.
PUB DATE May 83
GRANT USDA-ARS-NC-03713
NOTE 18p.; Paper presented at the Annual Meeting of the American College of Sports Medicine (30th, Montreal, Canada, May 1983).
PUB TYPE Reports - Research/Technical (143) -- Speeches/Conference Papers (150)

EDRS PRICE MF01/PC01 Plus Postage.
DESCRIPTORS *Biochemistry; *Body Weight; *Exercise Physiology; Human Body; Jogging; Males; *Metabolism; *Obesity
IDENTIFIERS *Somatomedin C

ABSTRACT

The growth hormone is a lipolytic hormone and somatomedin C mediates the metabolic effects of the growth hormone in many tissues. Growth hormone plasma levels are often depressed in obese individuals, and this low plasma level has been postulated as a reason for perpetuation of excess weight. Substantial weight loss in obese subjects improves growth hormone response to provocative stimuli. The present study investigated the effects of a 12-week progressive exercise program (consisting of jogging three times weekly) on growth hormone and somatomedin C plasma levels in five overweight males, 18-30 years of age and 15-40 percent above ideal body weight. Data collected at the beginning and end of the study included measurements for aerobic capacity, skinfold measures, body weight, fasting plasma growth hormone, glucose, and somatomedin C levels pretest and posttest. Mean body weight loss and decrease in skinfold measurement were significant during the program. Subjects' aerobic capacity increased. Changes in body weight accounted for 86 percent of the variability in somatomedin C changes; however, neither fasting plasma growth hormone levels nor growth hormone response to exercise stimulation changed significantly during the 12-week program. The responses suggest that somatomedin C is a more sensitive indicator than growth hormone of endocrine responses to exercise. Tables and figures with study results are appended. (Author/JD)

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The Influence of a 12-Week Conditioning Program on Growth Hormone and Somatomedin C Concentrations in Moderately Overweight Males[1,2]

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[1]Presented at the 30th Annual Meeting of the American College of Sports Medicine, Montreal, Canada, May 1983.

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ABSTRACT

Growth hormone is a lipolytic hormone and somatomedin C mediates the metabolic effects of growth hormone in many tissues. Growth hormone plasma levels are often depressed in obese individuals and this low plasma level has been postulated as a reason for perpetuation of the excess weight. Substantial weight loss in obese subjects improves growth hormone response to provocative stimuli. The present study investigated the effects of a 12-week progressive exercise program on growth hormone and somatomedin C plasma levels in five overweight males, 18-30 years of age and 15-40% above ideal body weight. Data collected at weeks 0 and 12 included VO_2max, skinfold measurements, body weight, and fasting plasma growth hormone, glucose, and somatomedin C levels pretest and posttest (Bruce Protocol). Mean body weight loss (11.4 lbs) was significant ($p < 0.05$) for the males during the program. A significant decrease ($p < 0.05$) in skinfolds occurred during the program. VO_2max increased from a mean of 38.8 ml/kg min to 44.6 ml/kg min (ns). Changes in body weight from week 0 to week 12 accounted for 86% of the variability in somatomedin C changes ($p < .05$). Neither fasting plasma growth hormone levels nor growth hormone response to exercise stimulation changed significantly during the 12-week program. The significant relationships between somatomedin C and body weight without significant relationships between growth hormone and body weight suggest that somatomedin C is a more sensitive indicator than growth hormone of endocrine responses to exercise.

Exercise may have a potential role in the normalization of endocrine and metabolic abnormalities present in obese individuals. Alterations in growth hormone and somatomedin C are of particular interest because of their function in energy substrate metabolism and storage, especially lipid metabolism and storage.

Growth hormone inhibits lipogenesis and promotes lipolysis in adipose tissue[8]. Some of the effects of growth hormone may be indirect since somatomedin mediates the effects of growth hormone on protein synthesis[13]. It is not clear whether somatomedin mediates the effects of growth hormone on fat metabolism since somatomedin does compete with insulin for binding sites in some tissues such as the liver[13].

Growth hormone is intricately involved in energy substrate metabolism through its effects on adipose tissue, muscle tissue, and carbohydrate metabolism. While growth hormone functions in regulating energy substrate metabolism, diet in turn regulates growth hormone levels, as evidenced by plasma alterations of this hormone during fasting situations and in protein malnutrition[14].

Somatomedins mediate growth hormone action in growth and development. To be considered a somatomedin, a substance must be under control of growth hormone, be insulin-like in its actions, and stimulate cell growth in one or more tissues[20]. The actions of somatomedins studied to date are anabolic on most body tissues. In muscle, somatomedins increase protein synthesis, increase the transport and uptake of amino acids, increase glucose uptake, and increase incorporation of glucose in glycogen. In adipose tissue, somatomedins increase DNA synthesis, increase glucose oxidation,

decrease lipolysis, and increase lipid synthesis[13, 16, 18].

Since by definition somatomedin is under control of growth hormone, there should be some type of correlation between growth hormone plasma levels and somatomedin plasma levels. In general, somatomedin plasma levels mimic growth hormone plasma levels. Furthermore, growth hormone exerts the most powerful influence over somatomedin plasma levels[10, 19]. However, during obese states, growth hormone levels are suppressed while somatomedin levels are normal or increased[3, 4]. The fact that alterations of growth hormone and somatomedin C in obese subjects are likely to be a consequence or a cause of increasing adiposity makes their evaluation prerequisite to the understanding of the implications of exercise.

METHODS

The purpose of this research was to compare the growth hormone and somatomedin responses of moderately obese, sedentary males to exercise stress tests at the beginning and end of a 12-week weight reduction program. The weight reduction program consisted of jogging 3 times weekly at 65-75 percent of maximum heart rate. Each activity session ranged from 20-60 minutes in duration (see Table 1). Nine males were recruited for the study. The data presented are based on 5 male subjects for whom complete data were available.

All subjects were screened for the absence of CHD, angina, diabetes and hypertension. Medical history was reviewed and informed consent was obtained for each participant. Participants were between 18-30 years of age and 15-40 percent overweight according to 1959 Metropolitan Life Insurance Company Standards[12].

Jogging therapy was conducted on a one-to-one basis by trained graduate research assistants and faculty. Prior to each jogging session, participants were required to complete stretching and warm-up activities for five to ten minutes. Stretching was done primarily to improve flexibility and reduce risk of injury.

Anthropometric measurements included height (cm), weight (lbs) and four skinfold measurements in mm (tricep, bicep, subscapular, suprailiac). Percent body fat was estimated from the sum of four skinfolds according to the equation of Durnin (1974) and quetelet index ($100 \times wt/ht^2$) was computed[7]. Two fasting blood samples, taken at the time of the treadmill test (pretest, posttest) were analyzed for growth hormone, somatomedin C, and glucose. Biochemical analyses for glucose were performed using the Technicon AA II System. Determinations of growth hormone and somatomedin C plasma concentrations were performed by radioimmunoassay. The treadmill test consisted of a continuous, grade-speed incremented exercise bout (Bruce Protocol). Each participant walked or ran until maximum fatigue. Measures of ventilation, temperature of expired gas, and percents of oxygen and carbon dioxide in the expired gas were obtained by the Douglas Bag Method of open circuit spirometry. The volume of expired air was measured and concentrations of oxygen and carbon dioxide in the expired air were determined using the Beckman OM-11 and LB-2 gas analyzers. Gas temperature was obtained from a thermistor inserted in the inlet port of the CD-4 gas meter. The method described by Consolazio, Johnson, and Pecora (1963) was used to compute O_2 uptake and CO_2 production[5]. The highest rate of O_2 comsumption obtained during the test was considered the maximal O_2 uptake.

Heart rate was determined from electrocardiograph recordings obtained during the last 10 seconds of each minute of the test by direct leads using a Beckman Type R411 Dynograph Recorder. The highest rate measured during the test was considered the maximal heart rate.

Simple regression was used to assess the effects of changes in weight, aerobic capacity (VO_2 max ml/kg min), quetelet and the sum of skinfolds over time on the changes in growth hormone, somatomedin C, and glucose. The acute responses to each treadmill test were evaluated by using the t-test for two related samples. The t-test for two related samples was also used to compare differences in growth hormone, somatomedin C, glucose, weight, aerobic capacity (VO_2 max ml/kg min), quetelet index, and the sum of skinfolds at weeks 0 and 12.

RESULTS

The results of the 12-week jogging therapy program are presented in tables 2 and 3. The mean body weight (lbs) and quetelet values for the five males at weeks 0 and 12 are presented in Table 2. Mean body weight loss (11.4 lbs.) from weeks 0 through 12 was significant ($p < 0.05$) for the five males used in the analysis.

Mean percent body fat and the mean of the sum of four skinfold value for the five males at weeks 0 and 12 are presented in Table 2. The t-test for related samples showed a significant decrease in skinfold measures from baseline at week 12 ($p < .05$). Percent body fat by estimation from skinfold measurements decreased significantly ($p < 0.05$) from weeks 0 through 12. The mean percent body fat decrease was 1.5 percent and the sum of four skinfold value decreased

from a mean value of 76.7 mm to 65.8 mm.

The t-test for related samples showed a non-significant increase in aerobic capacity from baseline to week 12 ($p < .10$). Aerobic capacity increased from a mean of 38.8 ml/kg min at week 0 to 44.6 ml/kg min at week 12.

Fasting plasma glucose concentrations are presented in Table 3. Significant pre- and postexercise fasting plasma glucose differences were observed. Postexercise fasting plasma glucose concentrations were significantly higher than preexercise levels at weeks 0 and 12 ($p < .005$).

Fasting plasma growth hormone concentrations, pre- and postexercise and somatomedin C concentrations, pre- and postexercise, are presented in Table 3. No significant changes in growth hormone concentrations or somatomedin C concentrations across time or between pre- and post-exercise concentrations were observed.

A significant ($p < 0.05$) inverse linear relationship between the change in preexercise fasting somatomedin C and the change in body weight (lbs) from weeks 0 to 12 was observed in the overweight males (Figure 1). Changes in body weight accounted for 86.1% of the variability in somatomedin C changes from week 0 to week 12. The regression equation stated that somatomedin C change = -1.488 - 0.137 weight change.

The quetelet index (wt/ht^2) provides an estimate of body composition and is frequently used in surveys[2]. This index is the best simple indicator of percent body fat for white men 19 years-of-age and older[6, 15]. A significant ($p < 0.05$) inverse linear relationship between the change in preexercise fasting somatomedin C and the change in quetelet index from week 0 to week 12 was observed in the overweight

males (Figure 2). Changes in quetelet from week 0 to week 12 accounted for 90.6 percent of the variability in somatomedin C changes during this period. The regression equation stated that somatomedin C change = -1.425 - 6.179 quetelet change.

DISCUSSION

Growth hormone responsiveness to exercise did not improve significantly in the male subjects even though significant decreases in percent body fat and body weight occurred during the program. The blunted responses of growth hormone to exercise after weight loss may be due to a delayed adaptation of hormonal mechanisms. Ball et al. (1972) found some obese individuals exhibited a blunted response to insulin-induced hypoglycemia up to six months after the achievement of ideal body weight[1]. The present study of 12 week duration may not have been long enough to observe improvements in growth hormone responsiveness to exercise. Also, the subjects did not achieve ideal body weight at completion of this study. El-Khodary et al. (1971) observed significantly less response of growth hormone to arginine infusion in those individuals who lost minimal amounts of weight as compared to those individuals who achieved ideal body weight[9].

The significant relationships between somatomedin C and body composition have not been previously reported in humans. However, Tannenbaum, Guyda, and Posner (1983) have reported that insulin-like growth factors such as somatomedin C play a role in appetite regulation, body weight regulation and growth hormone negative feedback regulation in rats[17]. Somatomedin C may be a more sensitive indicator than growth hormone of endocrine changes that may occur during weight loss with exercise. Londono et al. (1969) observed a linear relationship between change in growth hormone and change in body weight, similar to the

relationship we observed between somatomedin C and body weight, but weight loss was much greater (10-50 kg) than in this study[11]. The significant findings in this study relating changes in body weight to changes in somatomedin C but not changes in growth hormone, suggest that somatomedin C is a more sensitive indicator of endocrine adaptations.

Chaussain et al. (1978) investigated the effects of weight loss by dietary restriction on somatomedin activity in 18 obese children[4]. Mean weight loss for the children was 6.3 kg over a 3-week period with a 600 Kcal/day diet. Somatomedin activity for the 18 obese children was not statistically different before weight loss (1.07 ± 0.37 U/ml) than after weight loss (1.12 ± 0.36 U/ml) and no relationship between weight change and somatomedin change was noted. Growth hormone response to arginine infusion after weight loss in the obese children was significantly higher ($p < 0.05$) than baseline responses. The findings of Chaussain et al. (1978), compared with the findings in the present study suggest that hormonal changes related to somatomedin C and growth hormone with dietary restriction may be different than that of changes with an exercise program.

Recommendations for future research include the repetition of this study with analyses of growth hormone and somatomedin C for both males and females. Use of a control group for comparisons to the exercise group would help to determine those changes which occur due to the exercise conditioning versus those changes which occur from random fluctuation. In light of the findings of Tannenbaum et al. (1983), and of this study, further research is needed to investigate the role of somatomedin C in exercise adaptation, appetite regulation and weight loss in overweight individuals.

Acknowledgements

The authors would like to thank Drs. Louis E. Underwood and A. Joseph D'Ercole of the Department of Pediatric Endocrinology at the University of North Carolina at Chapel Hill for radioimmunoassay of growth hormone and somatomedin C.

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Table 1

Jogging Program Schedule for Weight Reduction[1]

week	Schedule	Frequency	Time
1-4	walk-jog	3 x / week	20 min
5-8	walk-jog	3 x / week	40 min
9-12	walk-jog	3 x / week	60 min

[1]Subjects exercised at approximately 65-75 percent of their maximum heart rate as determined by a maximum effort treadmill stress test (Bruce Protocol).

Table 2

Anthropometric Measurements During The 12-Week Program

	Week 0	Week 12
Weight (lbs)	197.2 ± 20.4	185.8 ± 19.1*
Quetelet (100 wt/ht^2)	4.06 ± 0.40	3.82 ± 0.37
Sum of Skinfolds[1]	76.7 ± 13.4	65.8 ± 13.2*
Percent Body Fat[2]	24.2 ± 2.2	22.7 ± 2.4*

[1]Skinfold measurements included bicep, tricep, subscapular, and suprailiac.

[2]Percent body fat was estimated from the sum of four skinfolds using the equation of Durnin[7].

*Significantly different from week 0 at $p < 0.05$.

Table 3

Fasting Plasma Glucose (mg%), Growth Hormone (ng/ml) and Somatomedin C (U/ml)

Week	Glucose		Growth Hormone		Somatomedin C	
	Pre	Post	Pre	Post	Pre	Post
0	102.6 ± 7.6	119.8 ± 13.3*	<1.0[a]	1.1 ± .40	1.48 ± .63	1.46 ± .60
12	98.2 ± 7.7	134.0 ± 27.1**	<1.0[a]	1.9 ± 2.1	1.55 ± .54	1.55 ± .54

[a]All preexercise growth hormone concentrations <1.0 ng/ml.

*Statistically different from preexercise at $p < .005$.

**Statistically different from preexercise at $p < .0005$.

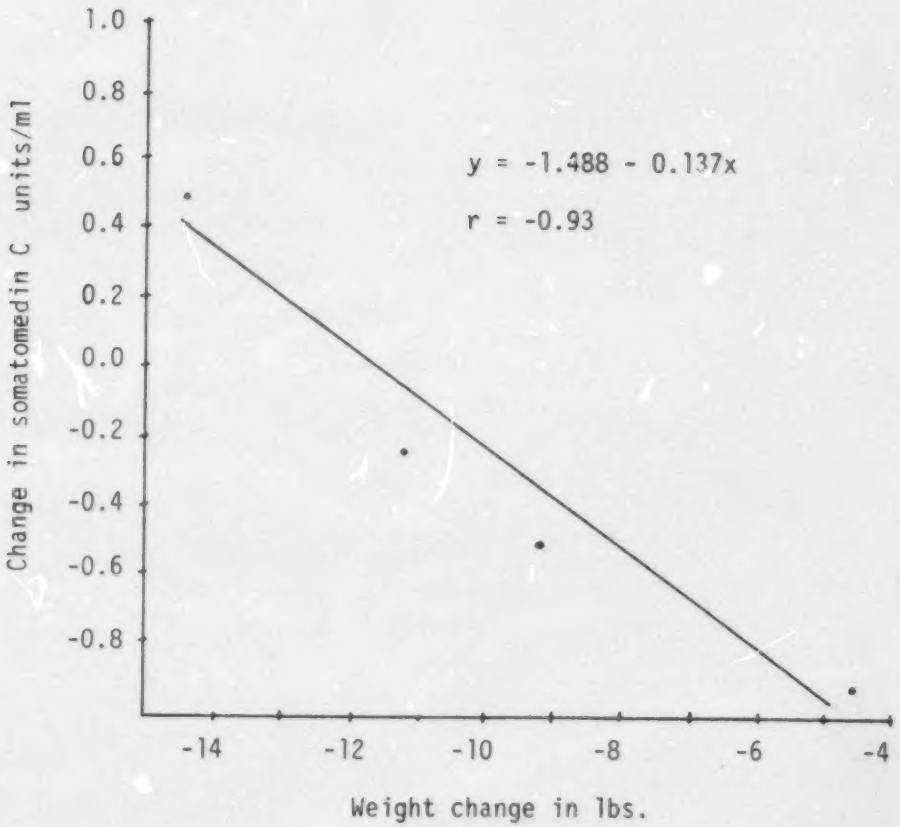

Figure 1. Effect of weight change on plasma somatomedin C. Each point represents changes from week 0 to week 12.

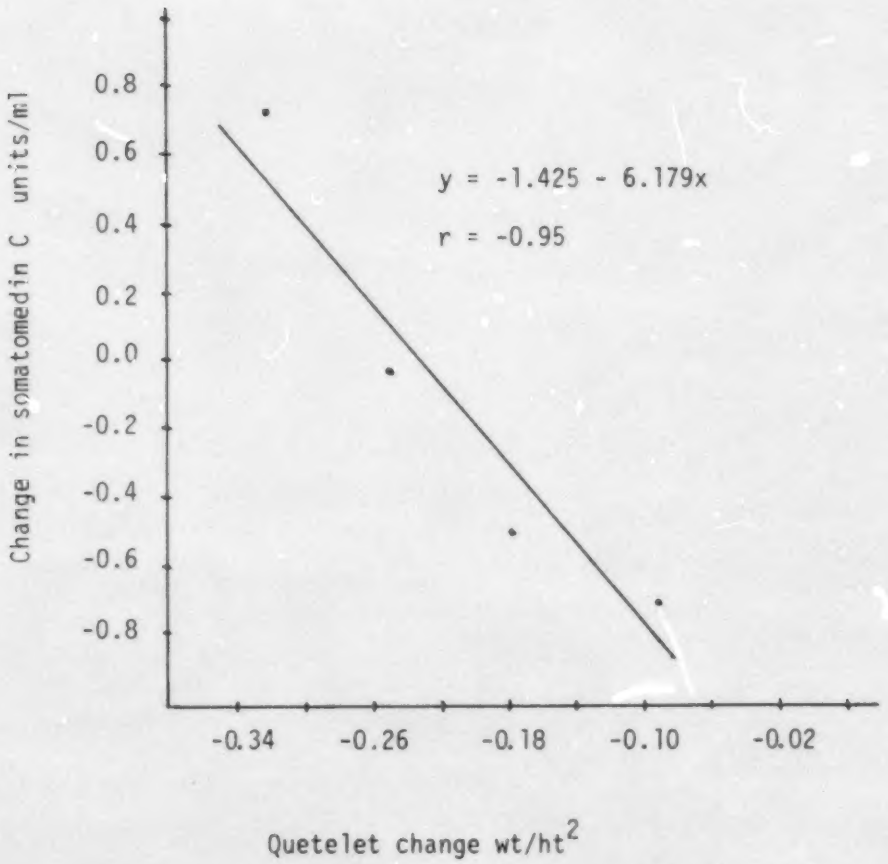

Figure 2. Effect of quetelet change on plasma somatomedin C. Each point represents changes from week 0 to week 12.